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Compliments of the Author.

TUBERCULAR LARYNGITIS.

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The subject which I have chosen is an old one, and not unnoticed in the records of the Association. (Vide Transactions of Missouri State Medical Association, 1875.) As it has been the theme of much discussion and contradiction I shall not attempt to review all that has been written by those who have maintained and disputed the existence of tuberculosis of the larynx, but will mainly endeavor to prove that the laryngitis so often associated with pulmonary phthisis is in many instances a tubercular infiltration. In presenting this subject, it is with full cognizance of the fact that much opposition has been offered to the views herein entertained, and that it has been denied by good authority that the larynx is ever invaded by tubercle. Let me add that the subject is an important one to the practical physician, for he will be influenced in his treatment accordingly as he believes this condition to be mainly a simple chronic inflammation or a local and characteristic part and parcel of a tubercular diathesis.

With your permission, then, I will (1) offer a definition of tuberculosis to which all will without doubt acceed, (2) show that the larynx is anatomically the place where, next to the lungs, we would expect to find tubercular infiltration, and (3) describe the characteristics of laryngeal complications as they are clinically and pathologically in harmony with the evidences of tubercle elsewhere.

(1.) The foundation of tuberculosis lies in a diminished vitality of the bioplasm, the germinal matter of the system. This condition inherited or acquired, favors the deposition of impoverished formative material in various localities, either as the result of inflammation, as at times in the lungs, or it may appear seemingly spontaneously in parts naturally designed for reception of healthy bioplasm. While inflammation plays an important rôle in phthisis, and in some cases is possibly independently of tubercle, the principal fretor of the disease,—yet the tubercle granula-

ton,—this infiltration of degraded bioplasm; the phthino-plasm of Williams constitutes the main ingredient in early phthisis. Wherever this material is deposited, unable to form tissue or sustain life, it clogs the circulation, irrititates the functions of the parts and so induces further change and degradation in itself and the surrounding st uctures.

The first lesion in pulmonary phthisis is tubercular infiltration of the angles where the bronchioles become continuous with the acini. (Rindfleish, Ziemssen's Cyclopedia, vol. v., p. 650.)

Virchow's description of tubercle is exceedingly lucid. At first the infiltration at these points in the lungs is pervaded by blood-vessels like other new formations, but its elements at length crowd so closely upon each other that the vessels become occluded, the corpuscles shrink, fatty metamorphoses occur, the fluid is absorbed, and the yellow cheesy degenerated mass remains. These observations, says Williams, prove grey tubercle to have a more definite organization than I was wont to ascribe to it in my earlier writings. This infiltration of degenerated bio plasts may occur not only in the adenoid tissue of the lungs, but wherever adenoid bodies naturally exist.

(2.) Impressed with the belief that pulmonary tuberculosis is the outgrowth of a prëexisting dyscrasia or diathesis, a local evidence of a constitutional fault, and that this evidence may be manifested wherever the structures are anatomically and pathologically favorable, 1 will attempt to show briefly that the larynx is by reason of its formation and function liable to tubercular deposit.

(*a*) In the larynx there exists the adenoid tissue shown by Heitler to be most abundant in the ary-epiglottic folds and over the arytenoid cartilages, the place where, in the larynx, the infiltrations are first noticed in phthisis. Add to this fact, the result of Klein's and Burdon-Sanderson's observations concerning the relation between the occurrence of tubercle and the adenoid tissue and the lymphadenoid nature of tubercle, and you have a strong point in favor of the existence of tubercle in the larynx.

(*b*) Tuberculosis in its local manifestations is mainly a disease of the respiratory tract. Not only does the larynx form a portion of this tract, but it is in such close relationship to the lungs that morbid processes may reach the larynx from the lungs by continuity of surface. Thus Von Ziemssen states that in the post mortem examinationt of phthisical patients he has repeatedly seen aphthous or flat ulcers, which Foster believes to be tubercular, extend by way of the bronchial tube and trachea to the larynx.

(*c*) Catarrh of the bronchial tubes may often be the exciting cause of tubercular disease in the lungs, and the larynx is not infrequently involved in, and is exceedingly liable to, the catarrhal inflammations. Other exciting causes may have an influence in

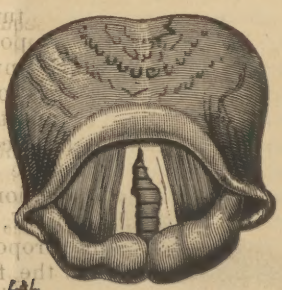
determining invasion in the larynx in tubercular subjects, such as the hyperæmia from excessive effort or the irritations induced by detrius from diseased lungs in contact with a sensitive laryngeal mucous membrane.

(3.) Premising then that the larynx is for the above reasons susceptible of tubercular invasion, we may concede Virchow's doctrine that the larynx is the best place to study tubercle, if in addition we find that the changes which do occur in the larynx in tuberculosis are characteristic. I do not mean that in all cases of laryngitis in phthisis we can find true tubercle, for there are many instances in which the catarrhal and follicular inflammations predominate, depending it may be on a tuberculous diathesis. The point in question is this: If pulmonary phthisis depends on tubercular infiltration and is a constitutional disease, then the typical laryngeal inflammation of phthisis, arising from like causes and in direct sympathy with the lung disease, is tubercular also, or as Flint (Phthisis, p. 127) states it, "both have a common causation." Indeed some of our best authorities (Tobold's Diseases of the Larynx, p. 124, Ziemssen's Cyclopedia, vol. VII, p. 833) limit the term laryngeal phthisis to the lesions of the larynx following pulmonary consumption and which are caused by miliary tubercles and tubercular inflammation.

One evidence of the identity of the nature of pulmonary and laryngeal phthisis is, that in a large proportion of the cases of pulmonary invasion, the larynx is also involved. Years ago Louis found among his recorded cases of phthisis, that 20 per cent. had ulceration of the larynx and there were, doubtless, many others that had not reached the ulcerative stage. In a series of one hundred cases which were examined carefully, regarding the frequency of this complication, I found the laryngeal disease more or less advanced in 57, and in a series of one hundred, recently completed, the proportion was nearly as large; ulceration was present in 24 in the first series, and in 31 in the second. Now grant that one person in five has laryngitis independently of lung disease—a large margin—there would remain 37 out of the 57, *i. e.*, 37 per cent. in the first series, in which it is but fair to conclude that the cause of the throat implication was in close relation to the pulmonary evil.

In regard to the stage of lung disease in which the larynx is first involved, it seems to be in some instances almost, if not quite as soon as the pulmonary invasion, but seldom precedes it, yet the appearance of the larynx even in incipient phthisis is often of value in making a diagnosis, where the other evidences are not conclusive. I have seen but four cases of laryngeal tuberculosis in which there was when first examined no evidence of lung involvement, though this soon followed in all. As two of these were well marked, and the sequalæ left no doubt of the diagnosis I will briefly refer to them.

W. A.————, æt. 35, of St. Louis. Merchant. Healthy in outward appearance and well formed. Was first seen January 4th, 1878. Gave this history: his father died of phthisis, but the rest of the famity were free from lung disease of any kind. His first illness was in July, 1877, when he lost appetite and strength, but had no cough, though his throat became sore. He regained his appetite, but his throat continued to trouble him and there was constantly "an aching sensation" in it. In November he became hoarse, and in December quite lost his voice. When I saw him in January, he had complete aphonia, and an irritating, hacking cough. Upon a laryngoscopic examination the vocal cords were slightly thickened and much ulcerated, especially at the processus vocalis, and also somewhat relaxed. The mucous membrane over the left arytenoid cartilage was infiltrated, and the left ary-epiglottic fold was greatly thickened. The corresponding parts of the other side were seemingly affected, but to a less extent. Upon careful examination, neither his attending physician, nor myself could find the slightest evidence of lung disease. A diagnosis of laryngeal tuberculosis was made. The patient was not seen again until May 5th; he was then spitting blood, and there was dullness and crepitation at the left apex with all the accompanying signs of phthisis.



[FIG. 1. Representing the larynx of W. A——, when first seen.]

Mr. D————, of Kansas, æt. 30; strong and vigorous, was first seen December, 1877. His father and mother both died from phthisis. His first illness was two years ago when his throat became sore, and he was hoarse and had an irritating cough; these symptoms became worse; for some time he was treated for syphlitic laryngitis, without effect. When examined there was thickening of the right arytenoid region, and the corresponding cord was invaded; the mucous membrane of the trachea, as far as could be seen, between the cords was inflamed; there were no ulcers; no evidence of lung disease; the diagnosis was the same as in the preceding case. He returned home and improved for a time, but on recent inquiry, I am informed that there is undoubted evidence of deposit in his right lung.

One of the first evidences of laryngeal implication is that even before there has been any noticeable infiltration, the mucous membrane may have a peculiar pallor, not the mottled appearance sometimes seen in simple follicular inflammation, but a general anæmia. In contrast to this there is often a dull red line of inflammation at points where there is the most vibration and motion. The hyperæmia from these and other causes results in local irritation, and the deposit of the degenerated plasma before referred to. This symptom occurs very early in the disease, and is often overlooked or neglected. The subjective symptoms at this stage are, inconstant hoarseness, dryness, and sometimes a tickling sensation, producing troublesome hacking cough. This cough is doubtless often due to reflex irritation of the laryngeal nerves, from disturbances of the pneumogastric by reason of the tubercular process in the lungs.

The laryngeal muscles may soon afterwards become involved, and their function impaired either by deterioration of the muscular fibres, or by the mechanical obstruction offered by the tubercular infiltration. The first, according to observations recently published by Frankël, is accomplished thus: There is first of all destruction of the transverse striæ of the muscular fibres, which may be reproduced by a fine-grained molecular (tubercular?) mass, or the nutrition and action of the fibrilæ are mechanically destroyed by compression consequent upon infiltration in the connective tissue. I believe also that this infiltration may so press upon the nerve fibrilæ as to further reduce the contractile effort of the muscles. In this condition and for the reason above given, the patient's voice will be weak, or become weak after continued effort. If the adductor muscles are affected, there will be at times more or less complete aphonia; if the tensors suffer there will be change in the vocal pitch or compass, and as both sets of muscles are generally implicated, both of these symptoms may be noticed.

The next step in the progress of tubercular laryngitis is the infiltration of portions of the larynx, especially in the regions of the arytenoid cartilage and ary-epiglottic folds. Tumefaction of the mucous membrane over the cartilages generally most intense on one side, may supervene, while the folds frequently assume a pyriform shape. The mucous membrane over the invaded parts is congested and distended, but not often actively inflamed. Sometimes in this stage there is evidence of follicular change and this has been erroneously exalted as the primary lesion. I believe with Schrotter, Gibb and others, (Chamberlain, Proceedings Conn. State Medical Association, 1876) that even these changes are tubercular when occurring in a tubercular subject. Wahlberg has found tubercular masses in these thickened parts where the follicles were normal, showing positively, that not only is this condition not the result of follicular inflammation, but may exist without it; when all the other parts of the larynx are more or

less diseased the follicles do not remain intact, but we must not mistake the effect for the cause. Where this typical inflammation is well marked, it will be generally found that pulmonary lesions also exist, and in the majority of cases in the lung of the side in which the larynx is most diseased. In this way the laryngoscope has became a valuable aid in the diagnosis of phthisis.

The pathological changes of this stage resemble those of tubercular deposits elsewhere. There is an infiltration of altered fibrinous exudation with a further development of small cells, with a single nucleus or of large cells, with multiple nuclei; these imperfect and irregular in shape, surrounded by a network of connective tissue, are crowded together in nodules or infiltrate the margins of the ulcers where the nodules are broken down.

There may be a sudden irruption and breaking down of miliary tubercles in the larynx, following the chronic tubercular infiltration, as described in illustration by Rindfleisch. These may break down and so ulcers occur. Miliary tubercle in the larynx has been well described by Tobold, "Chronic Diseases of the Larynx," p. 121, and by such authorities as Ter Maten, Turck, and Von Ziemssen, so that at this day it may scarcely be denied.

When the disease has advanced to ulceration we find, however, that in by far the greater number of cases, this is due to destructive change in the nodules crammed with cells of low vitality, and the gradual thinning and death of the mucous membrane. The character of an ulcer may generally be known by section and careful examination of its base. If we make a section through a laryngeal ulcer with the microscope, we may not find evidence of cheesy degeneration of the infiltrated mass, because the shallowness of the tissue in which the ulcers are, and the liability of the tubercles to break down, unless protected as they are, for example in the lung, forbid the change, but we do find, according to Rheiner, Hasse, Rindfleisch and others the unchanged tubercular granulations. An illustration of tubercular laryngitis, by Wahlberg, shows the infiltration of tubercular elements surrounded by bands of connective tissue at the base of the ulcers, the mucous membrane having been destroyed.

Ziemssen divides the laryngeal ulceration of phthisis into four classes. 1st. That depending upon true tubercle. 2nd. Follicular ulceration. 3rd. Specific infiltration of the sub-epithelial layer of the mucous membrane with cells and nuclei, and 4th. Apthous or erosive ulcers. It is interesting, however, to note that even in the three forms which he is indisposed to ascribe to tubercular changes, he is not wholly able to ignore tubercular influence. Of the follicular ulceration he follows Rindfleisch and says: "The primary condition in this form of phthisical ulcers, aside from the scrofulous or tubercular diathesis would be a purulent follicular catarrh, which only gives occasion to deeper alterations under the influence of that diathesis," *i. e.*, without the tu-

bercular element these deeper lesions do not occur. In discussing the 3d form, this author says the tubercular diathesis creates a vulnerability of tissue in which the specific infiltration excites inflammatory cell proliferation and subsequent caseation, and of the 4th form, the apthous, the statement is made that they may proceed from decaying miliary tubercles, while Forster (Pathological Anatomy) says most of the so-called apthous ulcers are tubercular.

I speak of this at some length, because these divisions embrace all the ulcers of the phthisical larynx, and yet each, including even the follicular ulceration of phthisis according to the author's own testimony, depends more or less closely upon a tubercular cause, while in two of the four divisions, the existence of true tubercle is readily admitted. The destruction of tissue progresses slowly, and may induce perichondritis, though usually before this is reached the patient succumbs to the advanced disease in the other organs.

The diagnosis of laryngeal ulcers in phthisis is comparatively easy if pulmonary lesions exist. Occasionally where the latter are slight, the laryngeal disease may be mistaken for either syphilis or carcinoma. The main points of differential diagnoses in the ulcerative stage of three diseases are approximately thus :

PHTHISIS.	SYPHILIS.	CARCINOMA,
First attacks the mucous membrane over the arytenoid cartiloges and the ary epiglottic folds.	First seen on the mucous membrane of the soft palate and walls of the pharynx.	First attacks the outer surface of the larynx and edge of the epiglottis. In rare cases may begin within the larynx.
Infiltration and destruction of tissue slow, and may remain stationary for some time.	Thickening and ulceration go on rapidly.	Progress of the disease slow but steady.
Borders of the ulcers ragged and worm-eaten in appearance. The outline of the arytenoids is lost, and the ary-epiglottic folds infiltrated.	Edges of the ulcers red, thickened and undermined —the thickening does not extend far beyond the border of the ulcer.	Ulcers have a dirty grey appearance. Edges raised and irregular.
Expectoration thin at first, changes to purulent with advance of the disease. From the laryngeal disease alone the discharge is seldom copious.	Expectoration thick, yellow, and with an offensive odor.	At first, scanty expectoration, but when ulceration is well determined, free and often mixed with blood and pus.
Voice weak and hoarse at times after exertion, and complete aphonia in the later stages.	Voice hoarse and rough from the beginning.	Voice often affected until the disease has made considerable advance.
Generally evidence of disease elsewhere, especially in the lungs.	Specific history and evidence of the disease in other parts of the body.	There may be no symptom of disease elsewhere.
Is most frequently developed between the ages of 25 and 50.	May occur at any age.	Is generally found after 50 years of age.

The general treatment of laryngeal tuberculosis must not be entered upon in this already lengthy article. But I would empha-

size this thought—the constitutional treatment is the main treatment, and he who loses sight of this cause, which lies far back of the local lesion in the symptoms produced by an ulcer or infiltration, be it here or there, fails to discharge his whole duty to his patient.

At the same time local treatment can not be well overlooked; indeed is generally necessary to secure comfort and to prolong life. In the very incipiency, where there is a depreciation of muscular power and anæmia of the larynx, I have probably had better results from the application of a solution of ferri et ammonia suph. than from any other. The effect of this as an astringent and local tonic is in direct variance with the tubercular development. Frequent gargles of weak solution of chlorate of potash may keep the larynx free from mucus and reflex irritation, but the patient must be taught to use the gargle so that the liquid may reach the larynx. The voice should not be used when it causes the least fatigue.

When the infiltration has progressed to any extent, anodyne applications may be used, or a hypodermic injection in the region of the larynx often gives relief. A strong solution of chromic acid carefully applied after the larynx has been freed from secretion has in my practice given decided relief in some very obstinate cases. This agent probably incites less irritation than the more commonly used caustics, and it is not as liable to produce ulceration as many of them are. The result is frequently a reduction of the thickening and of the hyper-sensitiveness of the mucous membrane.

After ulceration has begun, a happy temporary result may be attained by carefully touching the ulcer with a solution of hydrate of chloral, but care must be taken not to apply it indiscriminately to the parts. Counter irritation directly over the larynx seems to delay the progress of the ulcer in a number of cases. Elsberg has recommended trachæotomy, in cases where the larynx is greatly involved and the lungs in comparatively good condition, and this is often a question to consider.

But it is useless to catalogue the agents that have been used and abused in treating laryngeal phthisis. There is no definite line of treatment, for it must be remembered that the lesion here is but a symptom—an outgrowth, and must be treated as you would treat all such conditions—expectantly, and each case must be individualized.

And now, gentlemen, having taken up much of your time, let me say in conclusion, that I have endeavored to prove to you the existence of tubercule in the larynx, and to show how important a part it fills in laryngeal phthisis. I have referred frequently to authorities, for I have not wished to place before you statements, without foundation, at variance with views you may have entertained.

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